GYNAECOLOGY

Disorders of sexual development

Embryology of female genitourinary system

Cloaca → rectum, vesicourethral canal (bladder & urethra)
→ upper portion of cloaca
lower portion (urogenital sinus)

- pelvic part → lower urethra
- phallic part → testis / vagina

Homologous structures

Embryonal structure	♀	♂
• primordial germ cell	ova	spermatozoa
• genital ridge	ovary	testis
• sex cords	granulosa cells	seminiferous tubules, sertoli cells
• Gubernaculum	round ligament	gubernaculum testis
• Mesonephric tubules	epoophoron, paroophoron	efferent ductules, paradidymis
• Mesonephric ducts	gartners duct	epididymis, vas deferens, ejaculatory duct
• Paramesonephric duct	uterus, fallopian tube, upper vagina	prostatic utricle, appendix of testis, hydatid of Morgagni

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- SRY region is present on short arm of
  - → target gene for SRY & favour testis differentiation is SOX 9
  - → genes & favour ovary differentiation are WNT4, R3PO & DAX1

Gartner's cyst :

- It is a DD for UV prolapse
- vaginal epithelium over gartner's cyst is stretched & shiny, rugosities are lost } opposite in UV prolapse
- no cough impulse
- well defined
- ↳ - excision

Mullerian fusion defects :

Uterus didelphys

- paramesonephric duct fails to unite completely
- two horns of uterus & 2 cervix
- septum in vagina may be seen

Bicornuate uterus

- incomplete fusion of paramesonephric ducts < complete / partial

Septate uterus

- similar to bicornuate uterus, but no endometrial tissue in btw, only septum present < complete / partial

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Arcuate uterus:

- Mild indentation in fundus of uterus

Unicornuate uterus:

- One paramesonephric duct fails to develop
- only one horn present, other rudimentary horn
- may be communicating or non communicating
- ectopic pregnancy may occur in rudimentary horn

* Modality of choice for investigation → 3D USG
* surgery for bicornuate uterus → Strassman operation
  → $\bar{1}_x$ → recurrent abortion
* R_x for septate uterus → hysteroscopic septal resection

Mullerian agenesis / MRKH syndrome:

- 1° amenorrhoea
- 2° sexual characters well developed
- no uterus / cervix / vagina
- ovaries are developed
- repair for vaginal agenesis can be done at time of marriage → for coital purpose

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STD, PID, TB of Genital tract

STDs:

Bacteria	Viruses	Fungi	Protozoa	Ectoparasite
* Gonorrhoea	* HIV	* candida	* Trichomonas	* Scabies
* Syphilis	* HPV			* Pediculosis
* chlamydia	* HSV			
* LGV	* molluscum contagiosum			
* H. ducreyi				

Bacterial vaginosis:

- (N) flora ⇒ anaerobic >>> aerobic
  ↓
  eg: lactobacillus → maintains the acidic pH
- pH ⇒ 4 to 4.5 (N)
- vaginal cells produce glycogen under the influence of estrogen
- Lactobacillus acts on glycogen & produce lactic acid responsible for acidic pH
- When lactobacilli are outnumbered by gardnerella vaginalis, M. hominis, ureaplasma urealyticum, mobiluncus to cause bacterial vaginosis
- presents ∈ foul smelling, greyish white vaginal discharge
- profuse discharge

no itching (no inflammation)

Clue cells (X) are seen in microscopy

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- amine test → use 10% KOH → fishy odour in bacterial vaginosis
- AMSEL criteria is used to diagnose
- Not transmitted sexually
- RFs: → new sexual partner, many partners, douching, smoking, IUCDs.
- Rx → * no need to Rx the partner ✗
  * metronidazole 400 mg daily BD x 7 days ✗
- Whiff test is aka amine test
- abundance of gram variable coccobacilli may be seen
- discharge doesn't contain any cells, WBCs
- On ex → Cx is ✗ & no discharge

Candidiasis

- caused by C. albicans > C. glabrata > C. tropicalis
- occurs at pH < 4.5
- RF: immunocompromised states
- Sx → curdy white discharge
  - vulva is inflammed & red
  - severe itching
- Δ → 10% KOH → dimorphic nature of candida seen
- Types → complicated & uncomplicated

complicated	uncomplicated
• longer Rx duration	↳ • single episode, no RF
• > 4 episodes/yr	• easy to Rx
• ≥ 1 RF	• candida albicans
• non candida albicans inf	

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Rx :
- azole group of antifungals (clotrimazole, butaconazole, fluconazole)
  - for complicated → boric acid capsules

Trichomonas vaginitis

- caused by T. vaginalis
- sexually transmitted
- affects female >> male
- cause vaginal discharge, cervicitis
- Sx → dyspareunia, spotting, discharge, urethritis, proctitis
- males are asymptomatic
- discharge is frothy green & itching present
- Δ: swab + 10% KOH → motile organisms seen
- Speculum examination → strawberry cervix
- Rx →
  - metronidazole
  - Rx the partner too

Sequale

- Bacterial vaginosis → pregnancy : PTL, PPROM
  pelvic surgery : infection of cuff
  marker of other STDs

TB of genital tract:

- caused by M. tuberculosis (95%) & M. bovis (5%)
- m/c in childhood TB
- becomes symptomatic by puberty

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- Involve fallopian tube > endometrium > Cx > ovary > vagina & vulva
- Causes → infertility, ectopic pregnancy (fallopian tube dmg)
  → menorrhagia foll by amenorrhoea (endometrium)
  → ulcers (Cx, ovary, vagina)
- D → moth eaten appearance of tube on HSG
  aka beaded appearance / leopard spot appearance
  ± clubbed ends of fimbriae
- Rx → ATT (doesn't cure infertility)
  ↓
  golf club appearance
  - tobacco pouch appearance

PID:

- infection of upper genital tract
- causative → N. gonorrhoeae, Chlamydia trachomatis (m/c)
  (R) vaginal flora too

- D:

Minimum criteria → - uterine tenderness
- cervical motion tenderness
- adneral tenderness

Additional criteria → - white discharge PV
- Fever >101°f
- ↑CRP, ↑ESR
- NAAT (Dx gonorrhoea & chlamydia)

- Gonorrhoea → Sx develop in 3-7 days
  → cervicitis, urethritis, PID
- Chlamydia → no direct pathogenic effect on genital organs
  → presents ± sequale of PID
  → delayed hypersensitivity → damage of tubes → PID

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Rx:

NACO ← syndromic m/m → KIT ±
lower abd pain → KIT 6 → ceftriaxone, metro & doxy
↓ gonorrhoea ↓ BV ↓ chlamydia

- pt may present ± tubo-ovarian abscess → do laparoscopy, obtain cultures from fallopian tube
- most sensitive test for chlamydia → NAAT (PCR)
- DOC for chlamydia in pregnancy is erythromycin

Genital ulcers

- Syphilis → 1° chancre
  - → 14–21 days after sexual contact
  - → a single ulcer on vulva / vagina / cx
  - → painless ulcer ± raised indurated borders
  - → base of ulcer is clean & non infected ; deep
  - → disappears after some time
- H. ducreyi → cause chancroid
  - → red beefy, extremely painful ulcer
  - → occurs on vulva / vagina / cx
  - → single lesion
- Herpes, HSV 2 →
  - extremely painful, numerous ulcers
  - Hx previous episodes
  - occurs on vulva / vagina
  - occur in crops
  - Rx → acyclovir / ganciclovir → ↓ no of ulcers / recurrence
- LGV → buboes are present

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Sex & intersexuality

Puberty:

- 1st sign → thelarche (breast budding)
- 2nd → pubic/axillary hair → pubarche/adrenarche
- 3rd → menarche ≤ is just preceded by a growth spurt

* GnRH → pulsatile secretion (night > day) → responsible for pubertal growth in ♂

↓

LH >> FSH

↓

GnRH pulse throughout day

↓

FSH & LH → ovary → follicle development → estrogen

↓

priming of endometrium

- estrogen for 1 yr → 1° ovulation → progesterone → menarche

- precocious puberty → 2° sexual character before 8 yrs of age
- absence of 2° sexual characters & menstruation at 14 yrs

&

absence of menstruation even in presence of 2° sexual characters at 16 yrs

→ both needs evaluation i.e delayed puberty

Abn pubertal development

① precocious puberty (PP)
- central → aka gonadotrophin dependant precocious puberty
  - m/c is idiopathic
- peripheral

Central PP causes → hypothalamic hamartoma, optic glioma (in NF), radiation, head trauma, CNS anomalies, infection

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Peripheral PP causes → autonomous ovarian cyst, adrenal estrogen producing tumours, exogenous estrogen

* Hypothalamic hamartomas present ⊂ gelastic seizures / laughing fits

- precocious puberty → F > M
- delayed puberty → M > F
  → m/c is constitutional → hypogonadotropic hypogonadism
  → 1° gonadal failure : hypergonadotropic hypogonadism

McCune Albright syndrome :

- Precocious puberty
- Cafe au lait pigmentation
- Polyostotic fibrous dysplasia of bone
- somatic mutation in GNAS1 gene
- girls > boys

Basics of Ⓝ menstruation :

Ⓝ hypothalamus, pituitary, ovary / gonads, endometrium

Basics of gonadal sex :

Testis
- Sertoli cells → AMH → ⊖ mullerian hormone
  → Testosterone Binding Factor (TBF)
- Leydig cells → Testosterone, DHT → male ext. genitalia

De novo sex → ♀

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Classification of disorders of sexual development:

- Sex chromosome disorder:
  - 45 XO Turner
  - 47 XXY Klinefelter
- 46 XY disorder
  - pure gonadal dysgenesis (swyer syndrome)
  - partial gonadal dysgenesis
- 46 XX disorders

* m/c of ♀ 1° amenorrhoea → Turner > mullerian agenesis > testicular feminising syndrome

Cryptomenorrhea:

- D/t imperforate hymen / transverse vaginal septum / cervical atresia
- pt presents ∵ 1° amenorrhoea

Imperforate hymen

- 1° amenorrhoea, monthly abd. pain
- O/E → bulging bluish membrane
- USG → hematocolpos (blood in vagina), hematometra (blood in uterus)
  → blood may sometimes spill into abdominal cavity
- Rx → cruciate incision in hymen

Amenorrhoea

1°

2° → +ve progesterone challenge test

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- Kallman syndrome → • delayed menstruation / puberty + anosmia
  - defect in anosmin gene
  - defective hypothalamus
    → hypogonadotrophic hypogonadism
- Withdrawal bleeding following administration of progesterone indicates progesterone deficiency & (N) estrogen secretion
- Androgen insensitivity / testicular feminisation syndrome = (AIS/TFS)
  - 46 XY chromosome
  - though ~~so~~ testosterone is produced, testosterone receptor is absent
  - failure of testicular descent → inguinal swelling ±
  - external female genitalia
  - (N) breast development
  - absent pubic / axillary hair, 1° amenorrhoea

Reifenstein syndrome:

- almost like a lesser degree of AIS
- male ∴ some degree of feminization
- bifid scrotum, hypospadias
- partial androgen insensitivity syndrome

- Other syndromes ∴ delayed puberty → Prader Willi syndrome, Laurence Moon Biedl syndrome

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- Sheehan's syndrome :
  - Pituitary necrosis / apoplexy d/t severe ~~bag~~ PPH
  - 2° amenorrhoea

- 2° amenorrhoea is seen in persons ~~c~~ systemic diseases leading debility, anorexia nervosa etc. → It is hypogonadotrophic

- m/c ~~of~~ congenital adrenal hypoplasia → 21 OH deficiency

MCQs

- Fitz Hugh Curtis syndrome → violin string adhesions between (R) fallopian tube & undersurface of liver

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Menopause & HRT

- d/t loss of ovarian follicular activity
- cessation of ~~menopause for~~ menstruation for 12 months
- avg age ⇒ 45 to 55 yrs (50 yrs)
- before 40 → premature ovarian failure
- Menopausal transition → begins ↓ menstrual irregularity & extends to 1 yr after permanent cessation of menses
  → average of 4.7 years & lasts 4–7 years
- classification of female reproductive aging is called STRAW

Prediction of approaching menopause:

- Fall in the level of inhibin B → ↑ FSH
- Fall in anti mullerian hormone (AMH) suggest low ovarian reserve & low antral follicular count
- ↑ LH (but < FSH)
- Study of FSH level on ~~day~~ day 2-5 after last menstrual period detects premenopausal stage

Markers of ↓ ovarian reserve:

- Day 3 serum FSH > 10–15 IU/L
- day 3 serum estradiol > 60–80 pg/ml
- low AMH (0.2–0.7 ng/ml)
- ovarian volume
- low serum inhibin B
- antral follicular count (N–20 to 150)

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AMH

- AMH is secreted by sertoli cells (in ♂) & granulosa cells (in ♀)
- peptide hormone
- in ♂, AMH secreted by 7th week of IUL till puberty
- in ♀, AMH secreted after puberty
- (N) level is 2-6.8ng/ml
- Helps in follicular development & oocyte maturation
- Level reflects no. of growing follicles

In postmenopausal period:

→ ↑FSH, ↑LH, ↑ estrone

→ ↓ AMH, ↓ androstenedione

* No. of oocytes at menopause → 1000

Sx of menopause:

- menstrual Sx
- hot flushes
- dyspareunia
- ↓ed libido
- urinary stress incontinence
- osteoporosis
- neurological
- psychological

* Hot flushes → are d/t ↓ed estrogen & ↑ed noradrenaline

→ peripheral vasodilatation → skin temp ↑, esp in fingers & toes

↓

sweating, ↑ BP

→ d/t rapid fluctuation in estrogen levels

→ altered serotonin levels

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- Rate of bone loss after menopause is 3-5%

Perimenopausal osteoporosis:

- Bone mass measured by DEXA (dual energy x ray absorbimetry)
- gives i) T score (compares your results to a healthy young adult 20-35)
  - * expressed as std deviations from mean
  - * 0 score → (N)
  - * -1.5 to -2.4 → indicate osteopenia

ii) Z score → compares your results to a person of the same gender & age as yourself

- ** score -2.5 indicate osteoporosis

- all menopausal women should be given 1g Calcium (if not on HRT)
  - If on HRT ⇒ 500mg Calcium is given
- pH of vagina is maximum during menopause (pH 6-7.5)
- androstenedione is converted to estrone

* most potent estrogens is estradiol (E2) → reproductive age
  estrone (E1) → menopause [potency → E2 > E1 > E3]
  estriol (E3) → pregnancy (least potent)
- There is 50% reduction in androgen production & 66% reduction in estrogen at menopause

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- estrogen level → 10 to 80 pg/ml
  - estrogen level >40 pg/mL exerts bone & cardiotrophic effect & <20 pg/mL predispose to osteoporosis & IHD
  - Sr. FSH >40 is diagnostic of menopause

HRT

- Ix → vasomotor Sx,
  vaginal atrophy,
  osteoporosis prevention or Rx
  - re-evaluation of need for HRT at 6-12 month intervals
  - prescribed in lowest possible dose for shortest period
  - HRT is not useful in CAD
- Estrogen causes → ↓ LDL, ↓ cholesterol, ↑ HDL, ↑ TGs, ↑ coagulation factors, ↑ lithogenicity of bile (↓ stone forming capacity)
- progesterone causes → ↑ LDL, ↓ HDL

Drugs for postmenopausal osteoporosis:

- Antiresorptive agents:
  - estrogen
  - SERM - raloxifene
  - Bisphosphanates
  - Denosumab → mab against RANK-L
  - Calcitonin
  - vit D
- Anabolic agents (stimulate bone formation)
  - Teriparatide (recombinant human parathormone)

[Raloxifene → for bone, tamoxifen → for breast]

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- Raloxifene → ↑ incidence of venous thromboembolism
  - * ↓ # rate,
  - * doesn't cause endometrial hyperplasia / breast carcinoma
  - * S/E → hot flushes
  - * C/I in renal disease

* progesterone is added to estrogen in HRT only if uterus is present to prevent endometrial cancer ; ∴ not gn in pts who have undergone hysterectomy

* Hot flushes

Rx → hormonal agents

- estrogen - CEE, 17 βE2
- progestins - MPA
- combined E & P
- TSEC : CEE + BZA

[conjugated equine estrogen - CEE
Tissue selective estrogen complex - TSEC
Bazedoxifene - BZA]

Non hormonal

- SSRIs
- SNRIs
- clonidine
- gabapentine

C/I for HRT

- Thrombosis
- ~~Rest~~
- undiagnosed ab(R) genital bleeding
- Know / suspected / Hx of Ca breast
- Any estrogen dependant neoplasia
- TE
- stroke / MI
- liver d/s

- DOC for ↓ libido - androgens
- HRT ↓es alzheimer, colorectal Ca

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Abnormal uterine bleeding

Characteristics of Ⓝ menstrual cycle

- Ⓝ length of cycle : 24 to 35 days
- avg " - 28 days (seen only in 15%)
- No. of days of bleeding : 2-7 d
- Avg. no. of days of " - 4 to 5 d
- Ⓝ blood loss : 5 - 80 ml
- Avg " " : 30 ml

Definitions in abⓃ uterine bleeding :

- Menorrhagia → cyclic bleeding at Ⓝ intervals; the bleeding is either excessive in amount (>80 ml) or duration (>7 days) or both
- Metrorrhagia → irregular, acyclic bleeding
- Oligomenorrhoea → occurs less frequently i.e more than 35 days apart
- Hypomenorrhea → scanty bleeding, last < 2 days

Causes of metrorrhagia

- Fibroid polyp
- Ca Cx & Ca endometrium
- urethral ~~cat~~ caruncle
- IUCD
- decubitus ulcers
- withdrawal bleeding in pill users

Endometrium
- superficial functional layer
  - Stratum compactum (superficial)
  - Stratum spongiosum (deep)
- deep basal layer

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- The superficial functional layer is hormone sensitive whereas deep basal layer is hormone insensitive & contain stem cells. After every menstruation, regeneration occurs from deep basal layer

AUB

Anatomical causes (PALM)
- Polyp
- Adenomas
- Leiomyoma < submucosal / others
- Malignancy & hyperplasia

Functional causes (COEIN)
- Coagulopathy
- Ovulatory dysfunction
- Endometrial
- Iatrogenic
- Not yet classified

Causes of AUB:

General causes
- Blood dyscrasia
- coagulopathy
- Thyroid dysfn
- genital TB

Pelvic causes
- PID, pelvic adhesions
- uterine fibroids
- endometrial hyperplasia
- adenomyosis
- feminizing tumor of ovary
- endometriosis
- pelvic congestion
- varicose veins in pelvis

Contraceptive use
- IUCD
- post tubal sterilization
- POPs

Hormonal DUB
- ovulatory - irregular ripening / shedding
- Anovulatory - resting endometrium - 80%
- metropathia hemorrhagica

* Halban disease → irregular shedding of corpus luteum

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Puberty menorrhagia

- Rule out coagulopathy, pelvic lesions, hypothyroidism
- m/c is anovulation > blood dyscrasias
- Rx of PCOS → Cyproterone acetate
- Rx of puberty menorrhagia → progesterone, OCPs, GnRH analogues
- m/c endometrial pattern in DUB is (N)

H/m of AUB

- Young women
  - * progestogens
  - + Combined OCPs
  - * NSAIDs
  - * Tranexemic acid
  - * GnRH analogues
- in older women who have completed family
  - * hysterectomy
  - * endometrial ablation

In AUB → 1st investigation is TVS
IOC is endometrial Bx

- m/c of postmenopausal bleeding
  - globally → Ca endometrium
  - India → Ca Cx

Dysmenorrhea – cramping pain during menstruation

- 1° → no pelvic pathology
- 2° → pelvic pathology + → eg. fibroids, adenomyosis, PID, endometriosis

- Spasmodic dysmenorrhea → cramping pain on 1st & 2nd day, most prevalent
- Congestive " → manifest as Trg pelvic pain
- Anovulatory bleeding is painless

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ENDOMETRIOSIS

- Theories of endometriosis :
  - * Sampson's retrograde menstruation theory — most accepted & oldest
  - * Meyer & Ivanoff's coelomic metaplasia theory
  - * Stem cell theory
  - * Lymphatic or vascular spread

Common sites of endometriosis in decreasing frequency

- ovary
- cul de sac (pouch of Douglas)
- uterosacral ligaments
- vermiform appendix
- rectosigmoid colon
- ~~abc~~ ureter
- urinary bladder

* Endometriosis doesn't occur in spleen
* Endometriosis is hormone dependant
* 1° method of Δ is laparoscopy, ± or without Bx or histologic Δ (gold std)

RF :

- infertility
- multiparity
- pelvic pain
- high socioeconomic status, late child birth
- low BMI
- white women > other races
- red hair

Sx

m/c Sx

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O/E :- retroverted & fixed uterus may be seen
- cobblestone feel of uterosacral ligament is seen

Scar endometriosis:
- Painful & tender swelling over the scar
- usually not a/w pelvic endometriosis
- can occur at episiotomy sites, hysterectomy/cesarean scar, myomectomy/tubectomy
- cannot be treated ī hormones
- scar should be excised
- implantation theory explain its entity

Classification:
- American society for reproductive medicine classification: (ASRM 4)

* Stage I - minimal
II - mild
III - moderate
IV - severe

* menorrhagia may be seen
* Pain in endometriosis is d/t PG in peritoneal fluid
* Depth of penetration is more related to Sx than spread lesions penetration >5mm are responsible for pain, dysmenorrhea & dyspareunia

Examination findings
- pelvic tenderness
- bluish or blackish spots in posterior fornix & are tender
- U/L or B/L chocolate cyst (adnexal mass) of varying sizes

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Laparoscopic appearance:

- Early lesions: papular & red vesicles filled ± hemorrhagic fluid ± surrounding flame like lesions
- with age → dark red, bluish or black cystic areas
- powder burnt areas are inactive old lesions scattered over pelvic peritoneum
- Healed areas are seen as small peritoneal defects (windows) or white patches

Chocolate cyst

- mostly B/L
- lined by columnar epithelium
- pseudoxanthoma cells are large macrophages or scavenge cells & brown colour is d/t hemosiderin in them

Rx:

M/m of pain

Drug Rx:

- OC
- Mirena IUCD
- progestogens
- androgens
- GnRH
- letrozole
- RU-486

Laparoscopy

- destruction by cautery, laser vaporization
- excision of cyst
- adhesiolysis
- presacral neurectomy
- LUNA (laparoscopic uterosacral N ablation)

Laparotomy

- incision of chocolate cyst & removal of lining
- salpingo-oophorectomy
- hysterectomy & U/L or B/L salpingo-oophorectomy
- excision of scar endometriosis

In Infertility: clomiphene, letrozole (high dose)

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- Danazol causes absence of liver enzymes

LNG IUCD – non contraceptive uses
(Mirena)

- Menorrhagia / DUB
- dysmenorrhoea
- premenstrual syndrome
- endometriosis
- adenomyosis
- leiomyoma
- endometrial hyperplasia
- endometrial Ca
- HRT

* lifespan of mirena → 5 yrs

Adenomyosis :

- It is ingrowth of endometrium both stromal & glandular components directly into myometrium

Etiology : vigorous curettage, repeated childbirth / excess estrogen

Cf :
- women above age 40, parous woman
- menorrhagia (70%), dysmenorrhoea (30%), urinary frequency, dyspareunia, infertility

M/m :
- surgery is TOC
- hormonal therapy is not much effective

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Leiomyoma uterus

- Commonest benign tumor of uterus & commonest benign solid tumor in female
- Histologically, composed of smooth muscle & fibrous connective tissue.
- each myoma is derived from smooth muscle cell rests, either from vessel wall or uterine musculature
- derived from single progenitor myocyte

Classification:

- Subserous
- Interstitial / intramural
- Submucous
- cervical
- intraligamentary

* m/c site → posterior wall of uterus
* m/c Sx → abN bleeding
* m/c degeneration → hyaline degeneration
* incidence of sarcomatous degeneration - < 0.5%
* intermenstrual bleeding - submucous fibroid

Leiomyoma subclassification

SM - submucosal → 0 - pedunculated intracavity
1 - <5% intramural
2 - ≥ 50% intramural

O - other → 3 → contacts endometrium; 100% intramural
4 - intramural

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5- subserosal ; ≥ 50% intramural

6- subserosal ~~pedunculated~~ <50% intramural

7- subserosal pedunculated

8- other (eg: cervical, parasitic)

* Broad ligament fibroid is related to ureter

RF:

- Age
- african american race
- sustained hyperestrogenic environment
  - anovulation
  - PCOS
  - obesity
  - ↑ BMI
  - early menarche
- Family history

Reduced risk:

- smoking
- combined OCPs (↓ risk / no risk)
- post menopausal state
- ↑ed parity

* Fibroids are mostly asymptomatic

Pregnancy & fibroids:

- incidence in pregnancy 2%
- 80% of fibroids ↓ in size / show no change in size
- when they ↑, it is rarely >25%
- Complications
  - * preterm labor
  - * abruption
  - * malpresentation
  - obstructed labour, CS delivery
  - PPH
  - 2nd Trimester abortion

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- often confused ī adneral mass, MRI can be done after T3

Red degeneration:

- occurs in large fibroid, mainly during 2nd half of pregnancy & puerperium
- cause is vascular (no infection)
- Raw beef appearance ī cystic spaces
- Fishy odour d/t fatty acids & colour is d/t hemolysed red cells & Hb
- ↓scope → evidences of necrosis present, vessels are thrombosed
- USG may be helpful, but not diagnostic
- ℞ → analgesics, conservative

Degeneration of fibroids:

① Calcareous degeneration – phosphates & carbonates of lime are deposited in periphery along course of vessels. Form "womb stone"

② Hyaline degeneration

③ Cystic "

④ Fatty "

⑤ Red "

Sx:

- asymptomatic (80-90%)
- bleeding
- pressure & pain → urinary frequency, incontinence, constipation, dysmenorrhea, dyspareunia, noncyclical pelvic pain, uteric obstruction & hydronephrosis
- infertility & pregnancy wastage

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- GnRH receptor agents
  - leuprolide acetate
  - goserelin, triptorelin, nafarelin
  - GnRH receptor antagonists: cetrorelix, ganirelix
- Non hormonal
  - tranexemic acid
  - anastrozole, letrozole
- Radiological interventions
  - uterine A embolization
  - Magnetic Resonance guided focused USG (MRgFUS or MR-HIFU)
- Laparoscopic
  - laparoscopic uterine A occlusion
  - laparoscopic myolysis

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PCOS

m/cc of hirsutism in young ♀ → Idiopathic

m/c pathological c/of hirsutism in young ♀ → PCOS

m/c c/o rapid onset hirsutism – testosterone producing tumour

PCOS → ↑ed androgen

↓ aromatisation

estrogen

–ve ; +ve feedback

FSH ; LH → tonically elevated [fluctuating LH level is req for ovulation]

↓ failure of ovulation

↓ reversal of FSH : LH ratio [N FSH : LH = 2 : 1]

↓ no corpus luteum

↓ progesterone → infertility

big follicles → polycystic

- continuous proliferation d/t estrogen ↑
  no secretory phase d/t ↓ progesterone → endometrial hyper proliferation

↓ ↳ inadequate blood supply

menometrorrhagia ←

PCOS

- Rotterdam criteria → 2/3 – PCOS
- Androgen excess PCOS society

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Rotterdam criteria

1) Hyperandrogenism → clinically / biochemically
2) Ovulatory dysfunction → amenorrhea / anovulation
3) USG features of PCOS →
   - ≥ 12 follicles 2–9 mm in one or both ovaries
   - ovarian volume of ≥ 10cc

2/3 is diagnostic of rotterdam criteria

AE-PCOS criteria

- hyperandrogenism
- ovarian dysfunction – clinical / USG

2/2 is diagnostic of PCOS

* AE-PCOS society is applicable in absence of androgenic tumour
* In PCOS, ovaries can be (N)

Hormones ↑ed in PCOS

1) androgen (testosterone, androstenedione, DHEA, DHEAS)
2) estrone
3) ↑ LH
4) ↑ prolactin
5) ↑ LDL / TGs

Hormones ↓ed in PCOS

- ↓ FSH
- ↓ progesterone
- ↓ SHBG (sex hormone binding globulin)
- ↓ HDL

Associated syndromes

- ~~HAIRAN~~ syndr → hyperandrogenism, insulin (R), acanthosis nigricans
- Metabolic X syndrome → abdominal obesity, TG (>150 mg/dl), HDL (<50 mg/dl), BP ≥ 140/90, diabetic status

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Features / RF

- obesity
- insulin ® / hyperinsulinemia
- hyperandrogenism

* insulin ↓es SHBG → hyperandrogenism
* insulin ® = $\frac{\text{Serum fasting glucose}}{\text{serum fasting insulin}}$
- Acanthosis nigricans is the marker of insulin ® in PCOS
- ∴ 2hr OGTT is done in all PCOS pt ⇒ 35% of pcos have impaired glucose tolerance & 10% have DM

- Hirsutism → modified ferriman gallway scoring
  → max score : 36
  score ≥8 - mild
  ≥15 - moderate to severe hirsutism

M/m of PCOS

- weight reduction
- low dose OCPs (for irregular periods)
- metformin (m/c Ad/E – GI upset
  dangerous Ad/E – lactic acidosis)
- ovulation induction using clomiphene citrate, synthetic LH & FSH

M/m of hirsutism:

- androgen in PCOS is LH dependant (LH stimulate theca cells to produce androgen)
  &
- low dose OCPs gn for hirsutism ē desogestril, gestodene & norgestimate
  (3rd gen progestogens)

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Genital fistula

- m/c of vesicovaginal fistula < globally → gynae surgery
  India → obstructed labour
- most appropriate method for collecting urine for culture in case of vesicovaginal fistula is - Foley's catheterisation
- ~~uteroveg~~ uterovesical fistula } may present ⊼ cyclical hematuria
  bladder endometriosis }

Vesicovaginal fistula

- presents ⊼ continuous dribbling of urine from vagina, no urination thro' urethra
- 3 swab test - middle cotton swab is wet ⊼ urine & dye
- IOC - cystourethroscopy
- M/m → Latzko repair
- Timing of repair - after 3 months in obstetric fistulae,
  post surgery → within 24 hours → immediate repair
  ↳ late recognition → 3 months later

Ureterovaginal fistula

- DI+ hysterectomy
- continous dribbling of urine from vagina & (N) filling of bladder
- 3 swab test - upper most cotton plug is wet ⊼ urine but not ⊼ dye
- Further evaluation - IV CECT in excretory phase / IVP
- M/m - ureteric reanastomosis to be carried out as early as possible

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Three swab test

- ureterovaginal fistula - upper most swab soaked ~ urine but not ~ dye. Lower 2 swabs remain dry
- vesicovaginal fistula - upper & lower swabs remain dry but middle swab stained ~ dye
- urethrovaginal fistula - upper two swabs dry, lower swab stained ~ dye

* chassar Moir technique → repair vesicovaginal fistula
* most useful investigation in vesicovaginal fistula → cystoscopy
* m/c site of obstetric injury leading to ureterovaginal fistula → below cardinal ligament where uterine artery crosses
* Cause of retention of urine in reproductive age group
  - → cervical fibroid
  - → retroverted grand uterus
  - → severe UTI
* Retroverted uterus
  - → may be a/w endometriosis, PID
  - → may be a/w infertility
  - → may cause menorrhagia
* Kegel's exercise
  - → should be started 24 hrs after delivery (once the pt is pain free)

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Pelvic organ prolapse

vaginal
- endopelvic fascial defect - (cause)
- m/m → age independant

uterocervical
- ligament defect
- m/m - age dependant

Vaginal prolapse

Delancey's vaginal support

level 1 → apex → uterosacral & Mackenrodt's
" 2 → middle 3rd → paravaginal attachment
- i) arcus tendinous fascial pelvis
- ii) " " rectovaginalis

" 3 → lower 3rd → perineal membrane, muscle & body

* Defect in level 1 support → enterocele, apical prolapse, descent of Cx, vault prolapse
" 2 support → cystocele & rectocele
" 3 " → gaping introitus, deficient or lax perineum, urethrocele

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Anterior vaginal prolapse

- cystocele → puboresical fascia defect
- urethrocele → pubourethral fascia defect

Posterior vaginal prolapse

- enterocele
- rectocele
  - Fascia of Denonvilliers defect
  - deficient perineum

Management :

cysto & urethrocele → anterior colporrhaphy

rectocele → posterior colpoperineorraphy

enterocele → Moschowitz surgery + posterior colpo perineoraphy
↓
(purse string suture of POD)

lax perineum / deficient perineum → perineoraphy
↓
interstial tightening surgery

Uterine prolapse

True ligament → Mackendrot's, uterosacral & pubocervical

False " → round, broad ligament, ovarian ligament, angulation & serosa

M/M

- < 40 yrs, infertility, recurrent pregnancy loss → sling surgery / cervicopexy
  - Shirodkar
  - Purandar
  - Khanna's sling

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Shirodkar → isthmus of Cx to sacrum → most prefered

Purandare → isthmus of Cx to rectal sheath → less % but more failure

Khanna's sling → Cx isthmus to ASIS

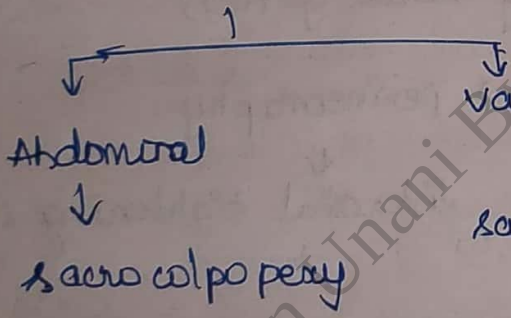
Shirodkar → more % but less failure

Khanna → ad/e → chronic periostitis

* >40, completed family → vaginal hysterectomy + vault suspension

* prolapse in pregnancy / puerperium → ring pessary

* vault suspension



(R) side preferred as in (L) side sigmoid colon may be damaged

* elderly, unfit for surgery → Le fort's surgery / colpocleisis (ring pessary is preferred)

* cervical elongation → Fothergill's or manchester operation

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Pelvic Organ Prolapse - Quantification system (POP-Q) classification

- 9 measurements ∴ hymen as reference point
- 3 x 3 grid

i) Aa → points on Anterior vagina 3cm proximal to external urethral meatus (midline)

ii) Ba → most dependant portion btw Aa & anterior fornix

Aa & Ba are marker of anterior vaginal prolapse

iii) Ap → posterior vagina midline 3cm proximal to hymen

iv) Bp → most dependant portion btw Ap & posterior fornix

(Ap & Bp - marker of post. vaginal prolapse)

v) C → Cx or vaginal cuff → marker of uterine prolapse of uterocervical length - ①

vi) D → apex of vagina / vault → uterosacral & Mackendrot's corresponds to

D is marker of enterocele

vii) Genital hiatus (GH) → distance btw external urethral meatus to posterior hymenal remnant

viii) Perineal body (PB) - posterior hymen to middle of anal opening

ix) Total vaginal length (TVL) → hymen to point D

Aa	Ba	C
GH	PB	TVL
Ap	Bp	D

⇒ 3 x 3 grid

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GH & PB → marker of tear perineum

$GH \propto \frac{1}{PB}$

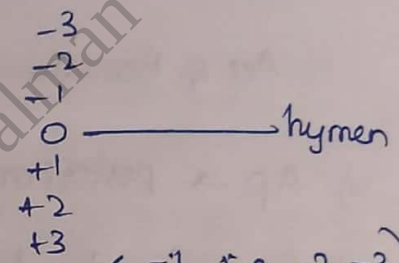
GH → (N) 4–5 cm ; TVL (N) → 9cm

PB → 3–4 cm

utero cervical length → (N) 3 inches

- All 9 measurements are done ⊼ full valsalva except TVL & is done after reducing the prolapse.

Staging



Type 0 → no demonstrable prolapse

1 → >1 cm above the hymen (i.e ● < -1 i.e -2, -3)

2 → ● -1 to +1

3 → more than stage 2, but 2 cm less than TVL

4 → ● more than TVL -2

Max measurement of Aa & Ba → +3

" " " Ap & Bp → +3

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Contraception

Classification

- Temporary → barriers, natural methods, pills, IUDs
- permanent → surgeries

Natural methods

i) Rhythm method

* calendar method (Ogino Knaus theory)
  * 1st unsafe day ⇒ length of shortest cycle – 20 days
  * last unsafe day ⇒ length of longest cycle – 10 days
* Temperature method → abstinence until 3rd day of rise of temp (temp. ↑ is d/t progesterone)
* Mucus method (Billing's method): abstinence on all days of noticeable mucus & for 3 days thereafter
* Symptothermal method: 1st day of abstinence predicted from calendar or mucus method. End of fertile period detected by BBT method

ii) withdrawal method (coitus interruptus)

iii) Breast feeding

iv) Persona - oestrone-3 glucuronide & LH testing in urine

v) Cycle beads

* Ectopic pregnancy may be a complication of calendar method

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Efficacy of contraception

Pregnancy rate per 100 women yr

- No method → 85
- natural → 25
- withdrawal → 27
- lactational amenorrhoea → 2
- condom, ♂ → 15
  ♀ → 21
- Implanon → 0.01
- vasectomy & tubectomy → 0.15
- norplant → 0.05
- Cu T 380 A → 0.8
- LNG 20 → 0.1
- combined OCPs - 0.1
- POP → 1
- DMPA & NET injectable → 0.3

Pearl index = $\frac{\text{no. of accidental pregnancies} \times 1200}{\text{no. of pt observed} \times \text{months of use}}$

↓

indicate contraceptive failure

Barrier methods:

- Condoms, spermicidal agents (nonoxynol-9).
- occlusive diaphragm → • dutch cap/diaphragm
  • cervical cap
  • female condom
  • today (CA sponge)

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female condom:

- prevents STDs

Diaphragm

- not suitable in pelvic organ prolapse
- proper sizing
- additional spermicide after each episode of coitus
- can cause TSS if left > 24 hours

Sponge (today)

- consist of a nonoxynol 9-impregnated polyurethran disc that can be inserted for upto 24 hrs prior to intercourse
- disc is moistened
- for efficacy, should be kept in place for 6hrs after intercourse & no longer than 30 hours

* contact dermatitis is m/c problem alw use of condom
* c/I of draphragm → recurrent UTI, uterine prolapse, herpes vaginitis
* Spermicidal jelly act by disruption of cell membrane

Combined hormonal contraceptives

- ethinyl estradiol (10-50 mcg) + progesterone
- mono / multiphasic
- MOA: * ⊖n of ovulation by suppression of release of FSH & LH estrogen suppress FSH & stabilise endometrium to prevent intermenstrual bleeding

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* progestins ⊖ ovulation by suppressing LH, thicken ⊕ mucus & renders endometrium unfavourable for implantation & fertilization

* uterine & tubal contractions may be modified to disfavour fertilization

* 3rd gen OCPs → contain norgestimate & gestodene ⊂ estrogens
→ more lipid friendly

Choice of progestrone

- 1st gen pills → ethinyl E2 + norethindrone
- 2nd gen " → " + norgestrel & LNG
- 3rd " → " + desogestrel, gestodene, norgestimate
- 4th " → ethinyl E2 + drospirenone

* Drospirenone → has antiandrogenic & antimineralocorticoid properties. useful in acne, premenstrual syndr, HTN in renal / adrenal insufficiency & hepatic dysfn. Can cause hyperkalemia

Non contraceptive benefits of OCPs

- ↓ endometrial & ovarian Ca
- fewer ectopic pregnancies
- more regular menses
- less salpingitis
- ↑ bone density
- less endometriosis
- ↓ benign breast disease
- probably less RA
- protection against atherosclerosis
- ↓ fibroids
- ↓ ovarian cysts
- ↓ PIDs

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- OCPs does not ↓ Ca Cx
- combined OCPs ↑ chlamydia & candida infections

C/I for OCPs (WHO MEC category 4)

- impaired liver fn
- Ca breast (known/suspected)
- ↑ TG / cholesterol
- undiagnosed vaginal bleeding
- smoker over 35 yrs
- past/present/family Hx of TE
- mod to severe HT
- diabetes ± vascular disease
- migraine ± aura

↓ amount of estrogen in Mala D is 30 mcg

Mala D ⇒ LNG 0.15 mg + ethinyl E2 30 mcg (21+7 iron tabs)

Drug interactions ± OCPs (Drugs ↓ing CO & efficacy)

- Rifampicin (at EE < 50 mcg)
- Griseofulvin
- phenytoin, phenobarbitone, primidone, carbamazepine, ethosuximide
- Tetra, doxy, penicillins, cipro, ofloxacin
- antiretrovirals

DMPA

MOA - ↑ Cx mucus viscosity, endometrium unfavourable for implantation & ⊖ ovulation (↓)

- 150 mg I.m every 90 days / 104 mg S.C
- Notable effects → * irregular vaginal bleeding
  * prolonged ovulation suppression after inj have been stopped
  * ↓ Bone mineral density
  * ↑ cervical carcinoma in situ
  * ↓ ovarian & endometrial Ca

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progesterone implants ↓ metrorrhagia

LNG-IUS (Mirena)

- T shaped polyethylene structure ī stem encased by a cylinder containing polydimethylsiloxane & LNG
- 52 mg LNG eluting 20 mcg daily
- approved for 5 yr use, but evidence for 7 yr
- Non contraceptive uses: ↓ menstrual blood loss, ↓ dysmenorrhea, ↓ PID, HRT in perimenopause
- ↓ menorrhagia

CuT

- Cu 380A approved for 10 yr use, but can be used upto 20 yr
- MOA - intense local inflammatory response induced in uterus by Cu containing devices leads to lysosomal activation & other inflammatory actions that are spermicidal.
  - ↓ fertilization occurs, inflammatory action against blastocyst.
  - endometrium becomes hostile for implantation
  - ↑ motility of tubes, induce endometrial atrophy

C/I for CuT:

- puerperal sepsis / postabortal sepsis
- unexplained vaginal bleeding
- GTN
- current PID/STD within past 3 months, known pelvic TB
- Ca Cx
- Ca endometrium

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- If pt becomes pregnant ⊼ IUCD, the IUCD should be removed to ↓ Rlo infection

Missing IUCD

- PV
  - ↓
  - UCG / erect Xray abd AP / lateral ⊼ sound
  - ↓
  - hysteroscopy
  - ↓
  - laparoscopy
- Emergency contraceptive doesn't interfere ⊼ early pregnancy
  - ↓
  - CuT, LNG & OCP's can be used
  - POP → LNG 0.75mg / LNG 1.5mg → ⊙ / delay ovulation
  - SPRM (Ulipristal acetate) - 30mg
  - COC - EE 0.1mg + LNG 1mg - Yuzpe method
  - CuT - 380A

* all are preferred within 72hr, can be used upto 120 hrs

Permanent methods

- Eligibility criteria
  - * married
  - * >22 & <49
  - * atleast one child >1yr age unless medically indicated
  - * female / her partner must not have undergone sterilization in past
  - * consent in sound state of mind

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♀ Sterilization

- Minilap methods → Pomeroy (used), Irving, uchida, fimbriectomy/ Kroener's tech, Parkland tech (used)
- Laparoscopy → Falope ring (m/c used), Hulka clemens clips, silicone titanium clip (fit & hire clip)

* ~~least method~~ effective method → vaginal fimbriectomy
* max chance of reversal → laparoscopic method = clip or ring
* tubes are ligated at isthmus
* reversal of tubal ligation → isthmo isthmic anastomosis
* No scalpel vasectomy is done now ; ~~test~~ involves ligation & division of spermatic cord ; partner may be on DMPA for 3 months
* Contraceptive implants → norplant, implanon, ~~[illegible]~~ jadelle

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Urinary incontinence & hyperprolactinemia

- (N) post. urethrovesical angle → 100°
- initial desire to void → 150 ml
  strong " → 250 ml
  residual → 50 ml
  bladder capacity → 400-600 ml
  flow rate → 15ml/sec
- intravesicle pressure → $10 cm^2 H_2O$
  intra urethral " → 20 - 50 $cm^2 H_2O$
  d/t urethral sphincter mechanism
- upper 2/3rd of bladder neck → above levator ani muscle
- urethral mobility mech → when pt coughs, pressure equally transmitted to bladder wall & upper 2/3rd blader neck
  ↓
  (70-75% of urinary continence)

Urinary incontinence

m/c → stress urinary incontinence (SUI) > detrusor instability urge incontinence

SUI

- involuntary passage of urine 2° to ↑ intra abd. pressure in absence of detrusor instability

Features →
- leak on cough
- small amount
- leak only till ↑ intra abd pressure
- no urge/ frequency/ nocturia / dysuria

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- other mech of SUI → * urethral mobility ~~mech~~ abN
  * intravesical pr > intraurethral pr
  * upper 2/3rd bladder neck below levator ani

- Causes : age, multiparity, obesity, chr cough, constipation, menopause, surgeries in bladder neck

Δ : clinical

diagnostic tests → i) stress test (pt in lithotomy position → cough ↓ sprinkling of urine)

ii) Q tip test → • done in empty bladder
• tipped swab introduced thro urethra & asked to cough
• when tipped swab move more than 30° → +ve test

iii) Bonney's test → 2 fingers in para urethral region & push up & ask to cough.
• if SUI corrected → +ve test

iv) Marchetty test → 2 allis forceps used instead of fingers

M/m of SUI

- Abdominal surgeries → Burch colposuspension, Marshall marchetty Kranz

Not done now ↓

Burch colposuspension	Marshall
→ proximal urethra/ • Cooper's ligament	bladder neck suspended • pubic bone • C/c - osteitis pubis

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- Vaginal surgeries

TVT (tension free vaginal tape)

- mid urethra is suspended on both sides
- both are day care surgeries
- muslin tape passed behind mid urethra (retrieve space entered where bladder is present, chance of injury +)
- cystoscopy must be done at end of procedure

TOT (transobturator tape)

- tape passed thro' both obturator foramen
- don't enter space of Retzius, no bladder injury

* TOT is the best method among all 4. (TOT > TVT)

Urge incontinence

- detrusor overactivity & parasympathetic overactivity

Features →
- frequency (>7 times/day)
- urgency
- nocturia
- dysuria

- M/c is UTI (∴ medical m/m)
- CNS causes should be ruled out (eg: parkinson, MS, alzheimers etc)

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Hyperprolactinemia

- structural similarity to GH & human chorionic somatomammotrophin
- $t_{1/2}$ → 20 mins
- ↑ed by coitus, sleep, nipple stimulation, pregnancy
- drugs ↑ing it ⇒ antipsychotics, dopamine antagonists, ∝ methyl dopa, verapamil
- pathological ↑ → pituitary adenoma, trauma, infections
- TRH → ↑ prolactin (∴ in hypothyroidism → prolactin ↑ed)
- hyperprolactinemia → head of amenorrhea, infertility & galactorrhoea
- in ♂ → erectile dysfn, hyperlipidemia, stroke, MI
- MOA of prolactin

→ prolactin has −ve feedback of GnRH

∴ ↓FSH & LH → no folliculogenesis → no ovulation

- Rx → bromocriptine is medical m/m
- Ⓝ ⇒ 15 to 25 ng/ml
- Protocols for ↑ prolactin

* 1st do UPT —+ve→ ANC clinic
  ↓−ve
* Rule out stress, sleep deprivation, drug intake
  ↓−ve
* Rule out hypothyroidism → Rx accordingly
  ↓−ve
* CT / MRI → pituitary adenoma → Rx accordingly
  ↓Ⓝ
  Repeat prolactin levels after 6 weeks

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Infertility

- 1 year is needed for Δ of infertility
- Type – 1° & 2°
- Infertility attributed to ♀ in 1/3rd, ♂ in 1/3rd & both in 1/3

Causes (↓ing order)

- Ovarian → anovulation, corpus luteum insufficiency
- Tubal → blockade d/t inf, surgery
- unexplained
- PCOD anovulation
- Uterine → Fibroid, synechiae, TB, malformations
- pelvic → endometriosis, adhesions
- cervical factor → lack of mucous, Abs in mucous

Testing

- Ovulatory dysfn → • ovulation predictor kit
  - early follicular FSH ± estradiol level (ovarian reserve)
  - ± AMH
  - ± serum TSH, prolactin, androgen
  - ± ovarian USG (antral follicle count)
- Tubal / pelvic disease → • HSG
  - laparoscopy + chromotubation
- Uterine factors → HSG
  - TVS / saline infusion sonography (SIS)
  - ± MRI
  - Hysteroscopy ± laparoscopy
- ♂ factor → semen analysis

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Asherman syndrome:

- Intrauterine adhesion / Asherman synd. is d/t destruction of endometrium in response to ovarian steroids
- Endometrial damage occurs d/t vigorous curettage following
  - PPH
  - miscarriage
  - elective abortion complicated by infection
- Δ: HSG / SIS
- Pt/m → hysteroscopy

* Best investigation for tubal patency → laparoscopic chromotubation

HSG

- Rubin's canula / foley's catheter is inserted into Cx & a radiopaque dye (usually 50% iodine + polyvinyl alcohol) is instilled into uterine cavity.
- performed on 9th / 10th day of cycle
- cannot diff btw cornual spasm & tubal block

Laparoscopic chromotubation

- done c diagnostic laparoscopy & a dye is injected thro Cx
- Free flow can be demonstrated at fimbrial ends
- Ix:
  - HSG showing abN finding
  - prior to planning tuboplasty
  - prior to IUI
  - prior to induction of labour

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- removal of hydrosalpinx prior to IVF
- PCOS to puncture the cyst to improve pregnancy rate of ART & avoid hyperstimulation syndrome
- suspected endometriosis

Sonosalpingography

- under USG, a Foley catheter is inserted, & 200 ml of saline injected into uterine cavity
- flow of saline along tube is visualized
- USG shows presence of free fluid in pouch of Douglas if tubes are patent

M/m of tubal factor infertility

- proximal (cornual) block - tubal cannulation / catheterization
- distal block - IVF, fimbrioplasty
- mid segmental block - resection & anastomosis
- Tubal recanalization
  - reversal best < clips > fallope rings > modified Pomeroy's > cautery
  - length of final tube : 4 cm
  - isthmo isthmic anastomosis has best results

Post coital test : (Cx mucous is examined)

- antisperm antibodies
- Cervical factor ab (N)
- penetration of sperm in Cx mucous - Miller Kurzrok test
- Sperm count & ab (N)

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Cervical factor infertility

- Abnormalities - cryosurgery / LEEP / conization
  cervical infection

(cervical mucous is permeable to sperm only during mid cycle)

- Evaluation - post coital test / Sims Huhner test
  - * couple asq to have intercourse on day of ovulation
  - * a subsequent sample of cervical mucous is ~~the~~ evaluated for elasticity (Spinnbarkeit) & no. of motile sperm per high power field

- M/m → intrauterine insemination

Male infertility

Pre testicular

- Endocrine deficiency
- psychosexual
- drugs → anti hypertensive, antipsychotics
- genetic → klinefelters, Y chr. deletion, single gene mutations

Testicular

- immotile cilia
- cryptorchidism
- infection
- torsion
- varicocele
- immunogenic
- 1° testicular failure

Post testicular

- obstruction of efferent duct:
  - cong. absence of vas deferens (cystic fibrosis)
  - acquired unf
  - surgery (vasectomy, herniorrhaphy)
- ejaculatory failure
- retrograde ejaculation
- hypospadias
- bladder neck surgery

* aspermia → complete absence of semen
* oligospermia / oligozoospermia → sperm count < 20 million / ml
* polyzoospermia → > 350 million / ml
* azoospermia → no sperm in semen

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- asthenozoospermia - reduced sperm motility
- leucocytospermia - ↑ white cells in semen
- necrozoospermia → spermatozoa are dead / motionless
- teratozoospermia → >70% sperm ± ab ♂ morphology
- oligoasthenoteratozoospermia → disturbance of all 3 variables

Semen analysis

- volume - >1.5 ml
- count - >15 million/ml
- Total motility >40%
- morphology >4%
- WBCs <1 million/ml
- Round cells <5 million/ml

In depth ♂ analysis

- serum FSH, LH, testosterone, prolactin, TSH
  - testicular dysfn → ↑ FSH & LH
  - ↓ FSH & LH → hypogonadotrophic hypogonadism
  - leydig cell dysfn → low testosterone & high LH level; elevated level d/t pituitary adenoma
- absence of fructose in seminal fluid → cong. absence of seminal vesicle or portion of ductal system / both
- Testicular Bx → Bx material is sent in Bouin's soln

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M/m of infertility

- ovulation induction
- IUI
- IVF - ET
- ICSI → intracytoplasmic sperm injection

ICSI

- Ix → severe oligospermia (5 million sperm/ml)
  - → astheno / teratospermia
  - → sperm Abs
  - → obstruction of efferent duct system
  - → cong. b/l absence of vas
  - → failure of fertilization in IVF

* Aspiration of sperm from testis is done by

i) Testicular sperm aspiration (TESA)

ii) " " extraction (TESE)

iii) Percutaneous epididymal aspiration (PESA) → trauma to epididymis may occur

iv) Microsurgical epididymal sperm aspiration (MESA)

Ix of IUI

* cervical factor infertility
* unexplained infertility
* oligoasthenospermia

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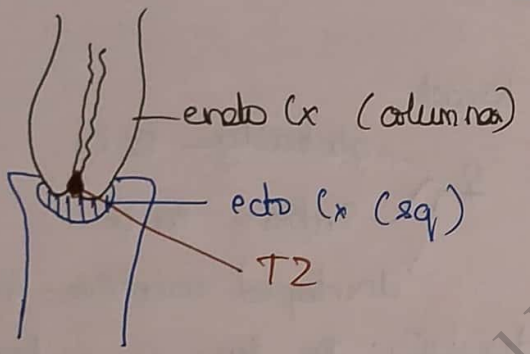
Ca Cervix

Anatomy of Cx:

- 2 parts → endocervix (part close to uterus) – columnar
  ectocervix → part projects into vagina, squamous epi
- squamocolumnar jn / transformation zone
- original SCJ is at external os, but rarely restricted to it



SCJ:

- endo Cx → red
  ecto Cx → pink
  T2 → white
- T2 moves out during pregnancy, puberty, OCP
  moves in into endo Cx ↑ age
- In all ♀, columnar epithelium of endo Cx changes to sq. epithelium of exo Cx. Thus metaplasia – physiological, in all ♀
- m/c site of Ca Cx – T2
- m/c variety of Ca Cx – sq. cell Ca
- as endo Cx is lined by columnar, adeno Ca of Cx can also occur
- m/c site for adeno Ca of Cx → endo Cx

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- m/c cell in menopausal ♀ → basal & parabasal cells
- In pregnancy & 2nd ½ of menstrual cycle → intermediate cells predominate
- in 1st half of menstrual cycle → superficial cells predominate

Superficial cells → large ~~eosinop~~ basophilic cell ± pyknotic nucleus

Intermediate cells → basophilic cells ± smudged outline

parabasal cells → basophilic, smaller cells ± large nucleus

Ca Cx :

- m/c Ca in Indian ♀ → Ca breast
- m/c Ca of genital tract in ♀ → globally – Ca Cx
  India – Ca Cx
  developed countries – Ca endometrium
- infection of Cx leads to dysplasia in few ♀ ± leads to
- Ca formation ± takes 10 to 30 yrs
- N:C ratio ↑ed in dysplastic cell

Classification

- < 1/3rd epithelium is dysplastic → CIN I
- > 1/3 but < 2/3 → CIN II
- > 2/3 → CIN III
- whole epithelium dysplastic but overlying epithelium intact – CIS
- overlying epithelium broken → invasive Ca

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Bethesda classification

- CIN 1 => LSIL (low grade squamous intraepithelial lesion
  2, 3 => HSIL
- CIS – no longer used

RF for CIN / Ca Cx:

- early age of intercourse
- " " " 1st childbirth
- multiparity
- low SES
- multiple partners
- all STDs
- smoking (RF for SCC)
- OCP (for adeno Ca Cx)
- HPV infection – most

* Early menarche & late menopause are RF for endometrial & ovarian Ca, not Ca Cx
- Ca Cx has no familial inheritance
- age group for CIN – 20-30 yrs
  CIS – 30-35 yrs
  invasive Ca – 35-39 yrs & 2nd peak at 5th to 6th decade

[endometrial & ovarian Ca are more common in nulliparous]

* all estrogen dependant conditions are more common in nulliparous women

* HPV affects basal epithelial cells (epitheliotropic)
  The cytological changes are called Koilocytosis
  Koilocyte → enlarged nucleus + perinuclear halo

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- High risk HPV → 16, 18 (70% cases)
  → 31, 33, 45, 52, 58 (30% cases)
- low risk → 6, 11 → genitals warts / condyloma accuminata
- Babies born to ♀ ~~with~~ genital warts have laryngeal papillomatosis, hence CS done

* viral protein responsible for malignant transformation
  - E6 & E7
  - E6 affects gene p53
    E7 " retinoblastoma gene
* viral proteins req for viral replication — E1 & E2
* m/c HPV a/w Ca Cx → HPV 16
  SCC Cx → HPV 16
  most sensitive HPV a/w ~~with~~ Ca Cx → HPV 16
  " specific " " " " " → HPV 18
  m/c HPV a/w adeno Ca Cx → HPV 18

Cancers by HPV

- ♀ → Ca Cx, vagina & vulva
  ♂ → oral, anal & penile Ca

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* HPV vaccines are made from inactivated capsid proteins

i) Cervirax → bivalent vaccine → protects against HPV 16,18 given to ♀ only

given to ♂ & ♀

ii) Gardasil → quadrivalent → 16, 18, 6, 11

iii) Gardasil-9 → 9 valent → 16, 18, 6, 11, 31, 33, 45, 52, 58

- Ideal age of vaccines → 11-12 yrs (9-26 yrs)
- most common adv E → syncopal attack
- ~~C/I~~ during pregnancy
- can be given to HIV +ve ♀
- protection rate of { gardasil → 75%, gardasil-9 → 90%.
- screening should continue after vaccine

Screening test → Pap smear

- start by 21 yrs
- universal screening, done 3yrly till 30 yrs
- and then if Pap test ~~[illegible]~~ +ve, HPV DNA testing - 5 yearly
  - only pap smear - 3yrly
  - HPV DNA testing in ♀ >30 yrs
- method - PCR / hybrid capture tech / southern blot
- screening continued ~~if~~ for 65 yrs if last 10 pap (N) till 75 if last 10 yrs pap suspicious

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- After hysterectomy for benign cause - stop screening
- In HIV +ve ♀, annual screening till 3 (N) results & then 3yrly screening
- Sampling instruments → Cx brush, cytobrush, plastic spatula, wooden spatulae (Ayer's spatula)
  - 1st slide → smear from T2
    2nd " → endocervix
    3rd slide → control slide → post. wall of vagina

* For hormonal study → lateral wall of vagina
  cytology → post. " " "

- Fixative used → 95% ethanol + 5% ether

* Liquid based cytology → done ⊼ Cervical brush
  → better than conventional pap
  → fixative is methanol

Alternative to Pap in rural areas

- VIA - visual inspection ⊼ 5% acetic acid - white stained areas ab (N)
- VILI → visual " ⊼ lugol's iodine - unstained yellow areas (stained area are bleak-brown) ab (N)
  - lugol's iodine - 5%
    schiller iodine - 0.3%

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- Pap smear detects – trichomonas, candida, HSV, HPV, actinomyces

doesn't detect – gonorrhoea, chlamydia

Result of pap smear

i) (N)

ii) Infection → Rx : ABx

iii) reactive changes

iv) atypical squamous cells of unknown significance – ASCUS

v) LSIL → enlarged, dark nuclei than ASCUS, ∴ less amount of cytoplasm

vi) HSIL – nuclei wrinkled & dark, very little cytoplasm

ASCUS → cells ~~[illegible]~~ ∴ slightly enlarged & darker nuclei compared to other (N) cells

ASCUS

- repeat pap after 6 months → ≥ ASCUS → colposcopy
- HPV DNA testing → +ve → colposcopy
- colposcopy directly. (better)

- 10% of LSIL → HSIL → (10%) → Ca Cx

LSIL

- visible lesion → punch Bx
- not visible → colposcopy directed Bx

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- HSIL → colposcopy directed Bx + endocervical curettage

* mild erosion of Cx & pap smear show dysplasia → ABx & repeat pap after 3 months

Colposcope:

- minimum magnification → 5 times
  max → 30°
- OPD procedure, no anesthesia
- disadvantage → cannot visualize endo Cx

Bx from any rough area / area appearing white before applying acetic acid - leukoplakia

↓

apply 3-5% acetic acid

↓

Bx from aceto white area → dlt dysplasia

↓

Bx from area c abN vessels

- Pap report → ASCUS / LSIL / HSIL

colposcopy report → CIN 1, CIN 2, CIN 3

- If papsmear → HSIL & colposcopy - N ⇒ cone Bx / conization done

Cone biopsy (conization)

- done under GA (OT procedure)
- Bx from ectocx, #2 & endocx

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Diagnostic Ix:

- unsatisfactory colposcopy
- discrepancy btw pap & colposcopy
- pap report is HSIL ± +ve endocervical curettage
- suspicion of microinvasion
- " " adenoCa

Therapeutic Ix:

- treat CIS or stage 1A1 of Ca Cx in young ♀

Lifecycle of CIN

	CIN 1	CIN 2	CIN 3
Regression	60	40	30
Persistence	30	35	50
progression to CIN II / CIS	10	20	
Progression to invasion	<1	5	20

CIN 1 → HPV DNA + pap smear yearly

↓

if persits ≥ 2 yrs

↓

cryosurgery

CIN 2 & CIN 3 → LLETZ (large loop excision of TZ)

OR LEEP (loop electro excisional procedure)

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Cryosurgery

- N_2O is passed at -20° temp → destroys cell
- m/c c/c → Cx discharge
- rare c/c → Cx stenosis & Cx incompetence
- never l/t cervical bleeding
- destroys epithelium upto 5mm deep

laser → " " " 7mm deep

- Rx CIN ≤ extends to vaginal fornices i.e CIN + VAIN

LEEP / LLETZ

- OPD procedure under LA
- diagnostic & therapeutic
- stainless steel wire used → thin coire + high power
- upto 10mm depth

Ix for hysterectomy in CIN

- recurrent CIN
- suspected adeno Ca & family of pt complete
- associated pelvic pathology for ≤ hysterectomy has to be done like fibroid
- pt cannot come for follow up

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Ca Cx

- m/c variety → SCC → * large cell keratinizing (m/c)
  - * " " non "
  - * small cell → worst prognosis
- adeno Ca → * m/c site is endo Cx
  - * seen in young ♀
  - * more common in OCP use
  - * a/w HPV 18
- m/c complaint in Ca Cx → irregular bleeding
  - most specific complaint → post coital bleeding.

m/m : * post coital bleeding only → pap smear → +ve → colposcopy

* post coital bleeding + growth → punch Bx

m/c in Ca Cx

- age - 35 - 39 yrs / 5th - 6th decade
- m/c route of spread - lymphatic
- site of met - obturator LN
- sentinel LN - paracervical / ~~iliac~~ ureteric LN
- m/c site of met in hematogenous spread - lungs

* sentinel LN assessment is very useful in → vulval carcinoma

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FIGO staging

- Investigation not recommended by FIGO → USG, MRI, CT, PET
- CT approved only to look for hydroureter
- Investigations like X ray, IVP etc are recommended by FIGO

Stage I → cancer confined to Cx

- Ia (microscopic cancer)
  - $I a_1$ ≤ 3mm deep
  - $I a_2$ 3–5 mm deep
- Ib (macroscopic cancer)
  - $I b_1$ < 4cms
  - $I b_2$ - ≥ 4 cms

Stage II - cancer involving upper 2/3 of vagina

II A - parametrium not involved

- II A
  - $II A_1$ - <4cm
  - $II A_2$ - >4cm

II B → parametrium involved

Stage III - Ca involving lower 1/3rd of vagina

III A - pelvic side wall ~~[illegible]~~ not involved

III B - pelvic sidewall involved / hydroureter / hydronephrosis

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Stage IV – Metastasis

IV A – local spread – bladder / bowel

B → distant met / inguinal LN involved

↓

only LN ± staging is changed

- Ca Cx rarely spreads to ovaries → no need of oophorectomy
- m/c of death in Ca Cx – uremia / renal failure
- ureter & kidney are involved in stage 3B of Ca, but bladder in stage 4A
- Most ⓧ prognostic marker in Ca Cx – stage > LN

Types of hysterectomy

	structures removed
① Simple / type I	~~total abdo~~ uterus + Cx
② Wertheims / type 2 / modified radical	→ uterus + Cx + 1 cm vagina + medial half of cardinal & utero sacral ligament + uterine artery after it has given branch to ureter + selective removal of enlarged LN
③ Type III / Meigs / radical	→ uterus + Cx + 2 cm vagina + whole of cardinal & utero sacral ligament + whole uterine artery + pelvic & para aortic lymphadenectomy

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④ Type IV → same like type 3 but upper 3/4th of vagina removed + periurethral tissue + superior vesical A

⑤ Type V (rarely done) → Type 4 + distal ureter & bladder also removed

* Highest R/o ureteric injury in c Wertheims hysterectomy
ureteric injury in m/c c simple hysterectomy

M/m of Ca Cx :

- all stages are radiosensitive, but Sy can be done only upto stage 2A.
- Stage 1A1 - 1B1 ⇒ Sy
  1A1 → conization / simple hysterectomy
  1A2 → wertheims
  1B1 → radical hysterectomy

Advantage of Sy - ovaries preserved, less vaginal fibrosis, coital fn retained

- m/c Sy done is radical / wertheims
- Radical trachelectomy - removal of 80% Cx, parametria, 1cm vaginal cuff c pelvic LN. later if the ♀ becomes pregnant, deliver by CS as vagina & uterus left are stitched

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- Radical trachelectomy is an option for women ī stage 1A1, 1A2 & 1B1 disease if Ca is <2cm in who desire uterine preservation & fertility

From stage 1B2 - IV

- Radiotherapy ī cisplatin as radiosensitizer
- Sb TOC - chemoradition
- CIS & stage 1a1 ī LVSI absent
  - Young ♀ → conization
  - old → simple hysterectomy (TAH + BSO)
- Stage 1a1 ī LVSI present & stage 1a2

* family not complete - radical trachelectomy & pelvic lymphadenectomy
* Sy - wertheims + pelvic lymphadenectomy (PL)

Stage 1B1

- Family complete → Type 3 / meigs (radical + PL & paraaortic sampling
- Not complete < tumor <2cm - trachelectomy
  big - hysterectomy

Postoperative m/m - done in all cases except stage 1A1

- If 3P's present → i) involvement of parametria
  ii) +ve LN
  iii) +ve margin

post op m/m of choice → chemoradiation

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- If 3S are present
  - large tumor size
  - stromal invasion > 1 cm
  - LVSI (lymph vascular space invasion)

post op m/m of choice → radiotherapy

Radiotherapy for Ca Cx

(A) Teletherapy (EBRT)

- go to ↓ size of tumor foll by brachytherapy
- Isotope used – cesium

(i) pelvic radiation –

(ii) extended field rdn includes pelvic & para aortic LN

(B) Brachytherapy

- Isotope → low dose rate tech – cesium 137
  high " " " – iridium 192

	Point A	Point B
location	2cm above & 2cm lateral to external os	2cm above & 5cm lateral to external os
structures present	paracervical / parametrial LN	obturator LN
Dose	early stage 80Gy to 85 Gy locally advanced → 85–90 Gy	50 – 55Gy → early stage 55 – 60Gy → late stage

- If i° Ca Rx by surgery, recurrent is Rx by chemoradiation
- If i° Ca ↓ chemo radiation → recurrent Ca
  - operable → pelvic exenteration
  - inoperable → palliative chemotherapy
    – cisplatin & paclitaxel

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Endometrial hyperplasia & Carcinomas

Endometrial hyperplasia

- Estrogen dependant (long term unopposed estrogen is most ⓧ cause)

Uterine causes of post menopausal bleeding (PMB):

i) endometrial atrophy (m/c)
ii) HRT
iii) endometrial polyps
iv) endometrial hyperplasia
v) endometrial Ca

RF: late menopause, multiparous, PCOS, obesity, DM, HT, tamoxifen, unopposed estrogen therapy, family history of breast, ovary, colon/endometrium, rdn menopause

→ corpus cancer syndrome

Types:

	progression to Ca (%)
• simple (cystic without atypia)	— 1
• complex (adenomatous without atypia)	— 3
• simple (cystic ± atypia)	— 8
• complex (adenomatous ± atypia)	— 29

Histopathology:

Simple hyperplasia

- glands large, cystic ± ↑ed gland to stromal ratio
- glands lined by columnar
- stroma - sparsely cellular
- scanty mitosis

Complex hyperplasia

- glands → ↑ed in size & no.
- overcrowding of glands
- pseudo stratified columnar
- stroma - densely cellular
- numerous mitosis

Characteristics of atypia

- cells large & irregular
- loss of polarity
- hyperchromatic nucleus ± prominent nucleoli
- altered nuclear cytoplasmic ratio

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Δ:

Sx → AUB or PMB

Δ → fractional curettage & histology

Rx: depends on age & type

Simple/complex hyperplasia without atypia

R/o progression to Ca in less

Young ♀ → ○ cyclic progestogen therapy for 6 months
- MPA for 21 days a month 3-6 months
- progesterone IUD

○ follow up after 6 months by endometrial sampling

peri & post menopausal → ○ cyclic/continuous progestin therapy or
○ hysterectomy

Simple/complex ± atypia: (R/o malignancy - high)

preferred Rx → hysterectomy Q

young women → • high dose progestin therapy (Megestrol) Q
• endometrial sampling every 6 months

Role of screening (not as effective as CIN)

methods → TVS, endometrial aspiration/Bx, vaginal pool smear

Endometrial carcinoma

• genital malignancy in developed countries → Ca endometrium > ovary > developing " → Ca cx > ovary > endometrium

• mostly in post menopausal (55-70 yrs)

• 10% ± PMB have Ca endometrium (EM)

RF: same as for endometrial hyperplasia + atypical EM hyperplasia

Protective factors: OCPs, progesterone therapy, multiparity, physical exercise

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Mode of spread:

- Direct spread → myometrium, serosa, adnexa, Cx, paraumetrium
- lymphatic " → occurs late to pelvic & para aortic nodes
- LN involvement is most (X) prognostic factor
  - inguinal nodes - involved in stage IV

CF:

- m/c Sx in perimenopausal → irregular vaginal bleeding
- " " post " → PMB
- watery offensive discharge → hydrorrhea / pyometra
- referred pain in hypogastric region — Sampson's pain
- BME - uterus atrophic / (N) size / enlarged

Δ:

- HPE of endometrium → diagnostic
- 1st inv in PMB → TVS

Features suggestive of Ca

- endometrial thickness ≥ 5mm (ET)
- fluid within cavity
- echogenic EM
- Ped vascularity

* ET < 5mm → review after 3 months / if bleeding recurs
* ET ≥ 5 mm → EM sampling

Methods for EM sampling

- EM aspiration - OPD procedure
- Fractional curettage → gold std
  → endocervical curettage foll by dilatation & EM curettage
- Hysteroscopic Bx → direct visualisation & targeted Bx
  → Ix : focal lesion / focal thickness

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- Pap smear → not reliable, +ve only in 10% Q

HPE types

- adeno Ca (endometrioid 80%) - m/c
- adeno squamous Ca - 15%
- papillary serous Ca (5-10%)
- mucinous adeno Ca (5%)
- clear cell adeno Ca (<5%) → most malignant & worst prognosis

Other inv → CT - myometrial invasion & LN met
MRI → best inv for "

Staging

- FIGO revised staging (2009)
- changes in all stages except stage IV
- surgico pathological staging

Stage I → tumor confined to uterus
IA → < 1/2 of myometrial invasion
IB → ≥ 1/2 of "

Stage II → invades cervical stroma, not beyond uterus
II → local and/or regional spread of tumor
IIA → invades serosa of uterus &/or adnexae
IIB → vaginal &/or parametrial involvement
IIIC → mets to pelvic &/or para aortic LN Q

Stage IV → involves bladder &/or bowel mucosa &/or distant mets
IVA → invasion of bladder &/or bowel mucosa
IVB → distant mets, including intra abdominal mets &/or inguinal LN Q

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Prognostic factors

- Most ⓐ in LN mets
- age ↑ed, stage of d/s, histologic type < adeno Ca → good prognosis / clear cell Ca → worst
- histological grade – grade III – poor prognosis
- myometrial involvement & extension to Cx → poor prognosis
- tumor size > 2cm → ↑ed LN mets
- hormone receptor status (+ve → better)
- ploidy status (aneuploid tumors → poor prognosis compared to diploid)
- HER-2/neu → poor prognosis
- +ve peritoneal cytology → controversial role (removed now)

Management

- Surgical staging
  - TAH + BSO + pelvic & para aortic LN sampling Q
  - clear cell & papillary serous Ca → omentectomy & peritoneal Bx in addition Q
- most pts Pd in early stages → Sy is 1st line Tx
- adjuvant RT following Sy to prevent recurrence Q

Tx for pelvic & para aortic LN sampling: Q

- clear cell, papillary serous, squamous Ca
- adeno Ca Gr III
- > 1/2 of myometrial invasion
- isthmic Cx extension
- extra uterine spread
- Tumor size > 2cm (only pelvic lymphadenectomy in absence of others)

Stagewise Rx:

- Endometrioid Ca stage Ia (Gr I & II) → TAH + BSO + pelvic & paraaortic LN sampling. NO post op therapy req

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- Endometrioid & stage Ia (Gr I), Ib → TAH + BSO + pelvic & para aortic LN dissection foll by post op RT
- Stage II → MRH (mod. rad. hysterectomy) + pelvic & para aortic LN dissection foll by post op RT
- III & IV → • debulking surgery foll by chemo ± RT or
  • chemo foll by RT or RT foll by Sy.

Choice of post op RT:

- Gr III → vaginal vault rdn
- adnexa, parametrial, pelvic LN inv → whole pelvis EBRT
- para aortic LN inv → extended field RT

Ct drugs: cisplatin, carboplatin, adriamycin, paclitaxel

Recurrent EM Ca

- m/c within 1st 2 yrs of Rx
- m/c sites of recurrence → vagina & pelvis
- m/c Sx " " → vaginal bleeding

M/m: • hormone receptor +ve → progesterone / tamoxifen
• RT for isolated recurrence
• If local therapy cannot be gn → palliative Ct

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Ovarian Tumours

Ovarian mass

- Non neoplastic & neoplastic

Non neoplastic or Functional cyst

Features :- related to temporary hormonal disturbances
- not >5-8 cm
- unilocular
- contains clear fluid
- asymptomatic
- usually regress spontaneously

Functional cysts → follicular cyst (m/c)
corpus luteal cyst
theca lutein & granulosa lutein cyst

Follicular cyst

- m/c
- d/t hyperestrogenism
- <5 cm
- single / multiple ; lined by granulosa cells
- p/w ~~PPH~~ EM hyperplasia & fibroid
- OCP prevent formation of new cyst

Corpus luteal cyst

- d/t overactivity of corpus luteum → hemorrhage inside corpus luteum
- may rupture → intraperitoneal hemorrhage (simulate ruptured ectopic)
- USG - cob web appearance
- may p/w amenorrhea / pregnancy
- spontaneous regression

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Lutein cyst

- a/w excess HCG like molar pregnancy, choriocarcinoma, clomiphene therapy
- B/L
- local pain
- conservative m/m

Ovarian tumours

- 2nd common genital malignancy
- 80% benign & 20% malign
- 90% malignant tumors are 1° & 10% are 2°
- 80% are epithelial ovarian tumours. 90% of ⅓ are benign
- 20% " non " " "

Classification

- epithelial ovarian tumours
- germ cell T
- sex cord stromal T

Epithelial tumors → benign / borderline malign / malign

- derived from surface epithelium
- serous (m/c) (75%), mucinous (10%), endometrioid (10%), brenner, clear cell, mixed epithelial

Sex cord stromal T:

- derived from ovarian stroma or mesenchyme
- secrete male or ♀ hormones
- granulosa cell, theca cell T → estrogen secreting
- sertoli cell, leydig cell T → androgen "
- gynandroblastomas

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Germ cell T:

- derived from primordial germ cells of ovary
- dysgerminoma, teratoma, yolk sac tumor, chorio Ca, embryonal Ca, polyembryoma

Benign ovarian T (ovarian cyst)

① Serous cystadenoma

- m/c benign epithelial tumor of ovary
- HPE - psammoma bodies
- arise from surface epithelium of ovary

② Mucinous cystadenoma

- arise from surface epithelium
- 10% B/L, 10% malignancy rest
- largest benign ovarian T
- cut section - honey comb pattern
- also pseudomyxoma peritonei

③ Dermoid cyst (teratoma)

- arise from germ cells
- m/c germ cell tumour
- m/c benign ovarian tumour
- m/c " " in reproductive age
- m/c " " " in pregnancy
- contain derivatives of ecto, endo & mesoderm
- m/c → ectoderm
- contain hair, teeth, bone, cartilage, neural & thyroid tissue
- Rokitansky protruberance - area of localised projection from s teeth, bone & hair project
- B/L → 15 to 20%
- R/o malign → 1 to 2%

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USG → • Rokitansky protuberance → hyperechoic nodule
• white ball app & dot and dash app → d/t hairs in cyst

X ray pelvis → teeth & calcification (radioopaque)

• SCC → m/c malignancy arising in dermoid cyst (1-2% risk)
• m/c c/c → torsion (mostly during pregnancy)
• struma ovarii → contain thyroid tissue
• Struma carcinoid → " serotonin & bradykinin from argentaffin cells

• Meigs synds - fibroma of ovary + ascitis + ® PE
• Pseudo meigs synds - ascites & PE occur ∈ Brenners tumor, thecoma & granulosa cell tumor
• Benign solid ovarian T → Brenner's, fibroma (m/c)

c/c of ovarian T:

• Torsion → dermoid cyst & serous cystadenoma
if gangrenous → ovariotomy
• intracystic hemorrhage → rupture d/t trauma / spontaneous / BME
• Infection
• pseudomyxoma peritonei → mucinous ascites 2° mucinous cystadenoma & cystadenoCa of ovary, mucocele of appendix / GB
→ poor prognosis
→ recurrence
→ Rx ⇒ TAH + BSO + removal of mucin, peritoneal implant & appendicectomy
• Malignancy (max in papillary serous cystadenoma & least in dermoid)

M/m of adnexal cyst:

• asymptomatic simple ovarian cysts < 5 cm → often physiological & resolve
• 5-8 cm, U/L, mobile, cystic → OCP, follow up in 3-6 months
if doesn't regress - Sy

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- cyst > 8cm, irregular, B/L, fixed, postmenopausal → further imaging & Sy.

Paraovarian cyst

- arise from vestigeal remnants of Wolffian duct in mesosalpinx or from peritoneal inclusion
- unilocular, contain clear fluid
- USG → ovary visualised separately from cyst
- always benign
- can undergo torsion
- Rx → enucleation of cyst, ovaries are preserved

Malignant ovarian tumours

- M/C → epithelial tumour
- M/C malignant " → serous cystadenoCa
- 5-10% hereditary predisposition
  - epithelial tumour → a/w p53 (aggressive & M/C gene) & K-Ras gene mutation

RF: 50-70yrs, early menarche, late menopause, obese, nulliparous, family Hx ovarian, breast, EM & colon Ca (hereditary ovarian cancer)

Protective factors: multiparity, Breast feeding, OCPs (used for 5yrs → 50% reduction)

* ~~Hereditary~~
* Familial ovarian cancer syndrome
  - i) Lynch II synd / HNPCC
  - ii) hereditary breast-ovarian cancer synd → a/w BRCA 1 (chr 17) & BRCA 2 (chr 13) → AD inheritance

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Malignant epithelial ovarian tumours

- C/F → • present in advanced stages ∵ non specific Sx
  - vague Sx → abd discomfort, bloating, dyspepsia, loss of wt & appetite

	Benign	Malign
• age	younger	extremes of age
• duration & progression	slow	fast
• cachectic Sx	–	+
• palpation	smooth, well defined border, cystic, mobile	irregular, ill defined border, variable consistency, restricted mobility
• site	U/L	B/L
• ascites	–	+
• POD deposits	–	±
USG	U/L	B/L
	unilocular	multilocular
	thin cyst wall	thick
	thin septations	thick
	anechoic	mixed echogenicity d/t solid areas
	no ascites	papillary projections, ascites, ↑RI
	high @ flow in Doppler	low @ flow pattern in Doppler

Risk malignancy index (RMI)

- calculated based on → USG features, menopausal status & CA 125 levels (multiply all 3)
- women ∈ RMI score >200 → refer to oncology centre

[Read index is for Ca Cx]

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Pattern of spread:

- M/C → exfoliation of cancer cells & implantation in peritoneal cavity (transcoelomic spread)
- less common — lymphatic & hematogenous

Δ: staging laparotomy is diagnostic

Tumor markers

- ↑ Ca 125 in non mucinous epithelial ovarian tumor (N < 35 U/ml)
  also elevated in PID, endometriosis, adenomyosis, genital TB, fibroid, endometrial Ca, colon, breast, pancreas, lung Ca

FIGO staging (surgical staging)

Stage I: Tumor confined to ovaries

- IA → limited to one ovary. No tumor on external surface, capsule intact & -ve peritoneal washings
- IB → both ovaries, no tumor on .... same as above
- IC → one/both ovaries ± tumor on ext. surface of ovaries / capsule rupture / malign ascites / +ve peritoneal washings

Stage II → Involve 1 or both ovaries ± pelvic extension

- IIA → extension to uterus / FT
- IIB → other pelvic tissues extension
- IIC → IIA / IIB ± tumor on ext surface of ovaries / capsule rupture / malign ascites / +ve peritoneal washings

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Stage III – one / both ovaries ± implants outside pelvis / +ve retroperitoneal nodes; superficial liver mets equals stage III

III A → histologically confirmed microscopic deposits of abd/peritoneal surface ± +ve retroperitoneal nodes

III B → macroscopic deposits ≤ 2cm

III C → >2cm

Stage IV → one / both ovaries ± distant mets

IV A → PE ± +ve cytology

IV B → parenchymal liver mets ± / or +ve inguinal nodes

Rx:

- Stage 1A, Gr I (low grade, low risk)

* surgical staging – TAH + BSO + pelvic & para aortic LN sampling ± infracolic omentectomy.
NO post op Rx req

* uterus & C/L ovary can be preserved in young women ± follow up

- Stage 1A (Gr II & III), stage 1B & 1C (high gr. high risk)

* surgical staging foll by post op ct

* chemo → cisplatin / carboplatin + paclitaxel – 6 cycles

- Stage II, III, IV → 1° cytoreductive / debulking Sy foll by post op chemo for 6 cycles

Optimum debulking / cytoreduction → when diameter of residual tumour nodules is < 1 cm

Neoadjuvant chemo → initial chemo foll by interval debulking Sy in pts in whom optimum debulking may not be possible

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Benefits of cytoreduction

- ↑ chemosensitivity
- improves immune fn

- Post ✂ surveillance – physical ex, CA-125, periodic CT scan
- 2nd look laparotomy → to determine response to therapy in pt ∵ no evidence of disease clinically
- 2° debulking → done for recurrence / persistent d/s after ✂

Progressive / persistent d/s : disease free interval < 6 months fall ✂

Recurrence : > 6 months interval

Other ✂ : intraperitoneal CT, immunotherapy (interferons & mab s like Herceptin), hormonal therapy → progesterones

- RT → only palliative

Dysgerminoma

- m/c malignant germ cell tumour
- corresponds to seminoma in ♂
- elastic rubbery consistency ∵ smooth capsule
- m/c malign. ovarian T alw pregnancy
- highly chemo & radiosensitive
- secretes placental ALP, LDH (+/-)
- malignant GCT ∴ best prognosis

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H/m of germ cell tumours (GCT)

- Sy → mainly
- U/L salpingo oophorectomy
- post op chemo → BEP regime – bleomycin, etoposide, cisplatin
- all cases needs post op chemo except stage IA dysgerminoma & stage IA gr I immature teratoma

Sex cord stromal tumours

- least common
- U/L
- m/c in perimenopausal age
- feminizing + → granulosa cell, theca cell T
- androgen secreting tumor – sertoli, leydig cell T

Granulosa cell tumors

- secretes estrogen
- Cf – children – precocious puberty
  adults – amenorrhea fol by prolonged bleeding
  PMB
- alw EM hyperplasia in 25–50%
- HPE – Call Exner bodies & coffee bean nucleus
- m/m → Sy → U/L SO in young
  - endometrial sampling
  - ~~Pelvic ex FT~~
  - post menopausal → TAH + BSO
  - post op chemo – BEP regime

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• Role of conservative Sy in young ♀:

→ U/L SO can be done in
- Stage I epithelial ovarian cancers (gr I)
- stage I GCT
- U/L sex cord stromal tumours

Metastatic tumors

- mets to ovary is mostly from 1° in GIT
- mostly B/L
- spread is transcoelomic or retrograde lymphatics

Krukenberg's tumor

- met from 1° in stomach > bowel > breast
- always B/L
- characteristic signet ring cells in HPE
- mets by retrograde lymphatics
- capsule intact, smooth, no adhesions, freely mobile, solid waxy consistency.

Tumor markers

- dysgerminoma — placental ALP, LDH
- yolk sac T — AFP, α1 antitrypsin
- embryonal Ca - HCG, AFP
- polyembryoma — HCG, AFP
- choro Ca — HCG
- Teratoma — —
- granulosa cell T - inhibin
- non mucinous epithelial G — CA 125
- mucinous " " — CEA, CA 19-9

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Histological characteristics

- Papillary cystadenoma – Psammoma bodies
- Brenner's – Walthard's cell nest (BMW)
- Clear cell T – Hobnail cells
- Dysgerminoma – large polygonal cells ± lymphocyte infiltration, fibrous septa
- Yolk sac T – Schiller Duval bodies
- Granulosa cell T – Call exner bodies, coffee bean nucleus
- Leydig cell T – Reinke's crystal
- Krukenberg tumour – signet ring cells

Borderline ovarian tumors (BOT) / low malign potential

- 30-50 yrs
- no stromal invasion
- mitotic figures < 4/10 HPF
- 90% survival rate
- serous type (m/c) > mucinous

M/m – staging laparotomy
- frozen section → If BOT +ve
  - * completed family – TAH + BSO
  - * not " " – U/L ovariotomy
- LN sampling not req

M/m in familial ovarian cancer

- genetic counselling, BRCA 1 & BRCA 2 genetic mapping
- Follow up ± TVS & CA 125
- OCPs
- family completed at / after 35 yrs → prophylactic SO (X) most (X) factor

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- annual mammography after 30 yrs
- OCPs for ≥ 5 yrs → ↓ risk by 50%
- 2 children + OCPs ≥ 5 yrs → ↓ risk by 70%
- prophylactic oophorectomy on family completion → only in familial ovarian cancers

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